

## Levels and profiles of organohalogenated contaminants in human blood from Egypt

Abdallah, Mohamed; Zaky, Amen Hamdy; Covaci, Adrian

DOI:

[10.1016/j.chemosphere.2017.02.139](https://doi.org/10.1016/j.chemosphere.2017.02.139)

License:

Creative Commons: Attribution-NonCommercial-NoDerivs (CC BY-NC-ND)

*Document Version*

Peer reviewed version

*Citation for published version (Harvard):*

Abdallah, M, Zaky, AH & Covaci, A 2017, 'Levels and profiles of organohalogenated contaminants in human blood from Egypt', *Chemosphere*, vol. 176, pp. 266-272. <https://doi.org/10.1016/j.chemosphere.2017.02.139>

[Link to publication on Research at Birmingham portal](#)

### General rights

Unless a licence is specified above, all rights (including copyright and moral rights) in this document are retained by the authors and/or the copyright holders. The express permission of the copyright holder must be obtained for any use of this material other than for purposes permitted by law.

- Users may freely distribute the URL that is used to identify this publication.
- Users may download and/or print one copy of the publication from the University of Birmingham research portal for the purpose of private study or non-commercial research.
- User may use extracts from the document in line with the concept of 'fair dealing' under the Copyright, Designs and Patents Act 1988 (?)
- Users may not further distribute the material nor use it for the purposes of commercial gain.

Where a licence is displayed above, please note the terms and conditions of the licence govern your use of this document.

When citing, please reference the published version.

### Take down policy

While the University of Birmingham exercises care and attention in making items available there are rare occasions when an item has been uploaded in error or has been deemed to be commercially or otherwise sensitive.

If you believe that this is the case for this document, please contact [UBIRA@lists.bham.ac.uk](mailto:UBIRA@lists.bham.ac.uk) providing details and we will remove access to the work immediately and investigate.

# Accepted Manuscript

Levels and profiles of organohalogenated contaminants in human blood from Egypt

Mohamed Abou-Elwafa Abdallah, Amen Hamdy Zaky, Adrian Covaci



PII: S0045-6535(17)30328-4

DOI: [10.1016/j.chemosphere.2017.02.139](https://doi.org/10.1016/j.chemosphere.2017.02.139)

Reference: CHEM 18902

To appear in: *ECSN*

Received Date: 19 September 2016

Revised Date: 4 February 2017

Accepted Date: 27 February 2017

Please cite this article as: Abdallah, M.A.-E., Zaky, A.H., Covaci, A., Levels and profiles of organohalogenated contaminants in human blood from Egypt, *Chemosphere* (2017), doi: 10.1016/j.chemosphere.2017.02.139.

This is a PDF file of an unedited manuscript that has been accepted for publication. As a service to our customers we are providing this early version of the manuscript. The manuscript will undergo copyediting, typesetting, and review of the resulting proof before it is published in its final form. Please note that during the production process errors may be discovered which could affect the content, and all legal disclaimers that apply to the journal pertain.

# **Levels and Profiles of Organohalogenated Contaminants in Human Blood from Egypt**

Mohamed Abou-Elwafa Abdallah<sup>1,2\*</sup>, Amen Hamdy Zaky<sup>3</sup> and Adrian Covaci<sup>4</sup>

<sup>1</sup>Division of Environmental Health and Risk Management,  
School of Geography, Earth, and Environmental Sciences,  
University of Birmingham,  
Birmingham, B15 2TT,  
United Kingdom.

<sup>2</sup>Department of Analytical Chemistry  
Faculty of Pharmacy, Assiut University  
71526 Assiut,  
Egypt

<sup>3</sup> Medical Oncology Department,  
South Egypt Cancer Institute, Assiut University,  
Assiut, Egypt

<sup>4</sup> Toxicological Center,  
University of Antwerp,  
Universiteitsplein 1  
2610 Wilrijk, Belgium

\* Corresponding author

Email [mae\\_abdallah@yahoo.co.uk](mailto:mae_abdallah@yahoo.co.uk)

Tel. +44121 414 5527

Fax. +44121 414 3078

## Abstract

Concentrations of polybrominated diphenyl ethers (PBDEs), polychlorinated biphenyls (PCBs) and some organochlorine pesticides (OCPs) were determined *-for the first time-* in serum of Egyptian colorectal cancer patients (n=35) and compared to a healthy control group (n=32). p,p'-DDE (the major metabolite of DDT) was the most frequently detected contaminant with the highest concentration (median = 131 ng/g lw) in all studied serum samples. BDE-209 was the least frequently detected contaminant with a median concentration <0.3 ng/g lw. The contamination profile in patients and controls was almost identical with p,p'-DDE showing the highest median contribution (77%) and oxychlordane the lowest (1%). The low p,p'-DDT/p,p'-DDE ratio (3.7%) in serum implies bioaccumulation and past exposure to DDT (c.f. recent and ongoing intake) . Statistical analysis revealed no significant differences ( $P>0.05$ ) between the levels of target contaminants in serum of patients and the control group. Gender, age and body mass index (BMI) were investigated as potential factors influencing serum contaminant levels.  $\Sigma$ DDT, hexachlorobenzene and pentachlorophenol concentrations showed significant positive associations with age and/or BMI of the participants. Comparison with other countries revealed concentrations of PBDEs and PCBs in Egyptian serum among the lowest reported worldwide.

**Keywords:** Organohalogen contaminants, OCPs, PBDEs, PCBs, Egyptian blood, Colorectal cancer

## Introduction

Organohalogen contaminants (OHCs) are a diverse group of chemicals including organochlorine pesticides (OCPs), polychlorinated biphenyls (PCBs) and polybrominated diphenyl ethers (PBDEs). Despite their various applications, these compounds are highly lipophilic, persistent in the environment and capable of bioaccumulation and biomagnification along the food chain.<sup>1, 2</sup> Moreover, numerous toxicological studies have associated different OHCs with adverse health effects including endocrine disruption, reproductive, developmental and neurological toxicity, type 2 diabetes and different types of cancer.<sup>3-6</sup> Combined with their ubiquitous distribution in almost all biotic and abiotic environmental compartments, these features have led to global concern over the production and use of OHCs. As a result, most OCPs, PCBs and PBDEs are currently listed as persistent organic pollutants (POPs) under the UNEP Stockholm Convention.<sup>1</sup>

While the regulatory actions have led to levelling off and even slight decline of the environmental concentrations of various OHCs in different regions, human exposure to these contaminants still occurs via direct and indirect pathways.<sup>7</sup> Several authors have documented the association between certain types of food (e.g. fish) and elevated serum levels of OCPs and PCBs,<sup>8, 9</sup> while others established significant positive correlations between PBDEs in indoor dust and their serum levels.<sup>10</sup> Recently, dermal uptake has been highlighted as a potential pathway of human exposure to PCBs and PBDEs.<sup>11</sup>

Due to their relevance for public health and associations with various diseases, several countries have collected extensive data on the occurrence and levels of OHCs in their population.<sup>12-15</sup> However, very little is known about the concentrations of OHCs in the blood of the Egyptian population. Furthermore, epidemiological studies have raised concerns over the increased incidence of colorectal cancer among the Egyptian

population, especially that patients under age 40 constitute 35.6% of all colorectal cancer cases in Egypt.<sup>16, 17</sup> Few authors have investigated genetic, dietary and life-style factors as potential determinants for the increased incidence of colorectal cancer in Egypt. Abdel-Rahman et al. reported an association between polymorphisms in the gene for the DNA repair enzyme XRCC1 with increased risk of colorectal cancer among Egyptians. Interestingly, an association between the studied polymorphisms and early age of disease onset was observed.<sup>18</sup> An epidemiological study of cancer of all organ sites in Gharbiah Province of Egypt from 1999 to 2002 revealed higher urban than rural incidences among both men and women for colorectal cancer.<sup>19</sup> This was linked to dietary preferences of the urban population with higher contribution of meat and fat as opposed to vegetables. Dietary risk factors were further highlighted by the results of another study from Minia governorate, where the most significant dietary and lifestyle colorectal cancer risk factors were higher consumption of red meat, preserved food, artificial sweeteners and fast foods.<sup>20</sup> However, the relationship between exposure to chemical environmental contaminants and colorectal cancer in Egypt is yet to be fully explored. This may be partly attributed to the lack of information on the levels of different OHCs in the Egyptian population. Therefore, the aims of the current study are: (a) to provide first insights into the occurrence and levels of PBDEs, PCBs in addition to some OCPs in serum of Egyptian colorectal cancer patients, (b) to compare the levels of target OHCs in serum of patients to an age- and sex- matched control group, (c) to investigate possible correlations between the levels of OHCs in patients and their age, sex or body mass index (BMI) and finally (d) to compare the levels of target OHCs in the Egyptian serum with those reported from other countries.

## Materials and Methods

### Target Compounds

The OHCs investigated in this study include:

- (a) Polychlorinated biphenyls (PCBs) no. 118, 138, 153, 170, 180.
- (b) Polybrominated diphenyl ethers (PBDEs) no. 47, 99, 100, 153, 154, 183, 209
- (c) Organochlorine pesticides, namely: pentachlorophenol (PCP), hexachlorobenzene (HCB), oxyhchlordane (OxC), 4,4'-dichlorodiphenyldichloroethylene (p,p'-DDE) and 4,4'-dichlorodiphenyltrichloroethane (p,p'-DDT).

### Reagents and chemicals

Solvents and chemicals used for analysis were of HPLC grade (Sigma-Aldrich, St. Louis, MO 63103 USA). Oasis® HLB SPE cartridges (6 mL/500 mg, 30 µm) were obtained from Waters (Milford, MA 01757 USA), while empty polypropylene cartridges (3 mL) and corresponding frits were purchased from Supelco (Bellefonte, PA, USA). Individual standards of OCPs and PCBs were obtained from Dr.Ehrenstorfer Laboratories (Augsburg, Germany), while PBDEs standards were purchased from Wellington Laboratories (Guelph, ON, Canada).

### Sample collection

Blood samples (5 mL, n=35) were collected from colorectal cancer patients admitted to the South Egypt Cancer Institute (SECI) between October and December 2012, while control samples (n=32) were obtained from the Upper Egypt Blood Bank from healthy donors at the same period. Informed consent was obtained from each participant following approval of the research protocol by the SECI ethics committee. Selection

criteria included Egyptian nationality, living in the Upper Egypt region for the last 8 years and lack of occupational exposure to any of the studied chemicals. Exclusion criteria were family history of colorectal cancer, patients with a primary cancer other than colorectal cancer and severely ill cases. The controls were healthy, unrelated to the patients, from the same geographical area and with no family history of colorectal cancer. Demographic information on the participants' age, sex and BMI are provided in table SI-1.

The serum was separated by centrifugation, transferred to clean tubes and kept frozen at  $-20^{\circ}\text{C}$  until analysis. Cholesterol and triglycerides were determined at the collection clinic using routine laboratory analysis then the value for total lipids (TL) was estimated according to the method of Phillips et al.<sup>21</sup>

### **Sample extraction and clean-up**

Sample extraction and clean-up were performed according to a previously reported method.<sup>22</sup> Briefly, thawed and homogenized serum samples ( $\sim 3$  mL) were spiked with internal standards mixture (BDE-77, BDE-128,  $^{13}\text{C}$ -BDE-209, PCB 143,  $\epsilon$ -HCH), then mixed with 2 mL of milliQ- $\text{H}_2\text{O}$  and 50  $\mu\text{L}$  of formic acid prior to ultrasonication for 20 min. The equilibrated samples were loaded onto pre-conditioned Oasis HLB cartridges, rinsed with 4 mL of milliQ- $\text{H}_2\text{O}$  and dried under vacuum. Target analytes were eluted with 10 mL of DCM:MeOH (4:1, v/v) and 2.5 mL of hexane. The eluate was dried under a gentle stream of  $\text{N}_2$  and resolubilized in 500  $\mu\text{L}$  of hexane:DCM (9:1, v/v).

The crude extract was cleaned-up over 800 mg of pre-washed silica topped with 100 mg of 10% acid silica packed in 3 mL polypropylene SPE cartridges. PCBs, PBDEs and DDTs were eluted first by 8 mL of hexane, followed by PCP in 10 mL of DCM. The 1<sup>st</sup> fraction was evaporated and redissolved in 80  $\mu\text{L}$  of isooctane containing 100 pg/ $\mu\text{L}$  of PCB129



and  $^{13}\text{C}$ -BDE-100 used as recovery determination (syringe) standards (RDS) for quality assurance/quality control (QA/QC) purposes. The 2<sup>nd</sup> fraction was derivatized by methylation and finally redissolved in 80  $\mu\text{L}$  of iso-octane containing 100  $\text{pg}/\mu\text{L}$  of PCB129 used as RDS. Further details are provided in the SI section.

### **Gas chromatography/mass spectrometry (GC/MS) analysis**

Instrumental analysis of target analytes was performed using a FOCUS GC coupled to a DSQII mass spectrometer (Thermo Fisher Scientific, Austin, TX, USA). Separation of target analytes was achieved on an Agilent DB-5 column (15m x 0.25mm; 0.1  $\mu\text{m}$ ). The mass spectrometer was run in selected ion monitoring (SIM) with ion source, quadrupole and mass transfer line temperatures set at 230, 150 and 300  $^{\circ}\text{C}$ , respectively. Further details of the GC/MS methods<sup>22</sup> are provided in the SI section.

### **QA/QC**

A multi-stage QA/QC protocol was adopted to investigate the accuracy and precision of the applied analytical method at different stages. Five-point calibration curves were created for all the analytes with a good linearity ( $R^2 \geq 0.986$ ) across the studied concentration range. None of the target compounds was above the detection limit in procedural blanks (n=8) analysed alongside the serum samples. Method accuracy and precision were evaluated via replicate analysis (n=5) of NIST SRM1958 (organic contaminant in human serum). Our results compared favourably with the certified concentrations of all target compounds (Figure SI-1) indicating good accuracy and precision of the applied method. Good recoveries (72-114%) were obtained for all the surrogate standards.

## Statistical analysis

Statistical analysis of the data was conducted using Excel (Microsoft Office 2010) and SPSS version 22.0. Statistical distribution within each dataset was evaluated using *Kolmogorov-Smirnov* goodness of fit tests. The results - combined with visual inspection of frequency diagrams - revealed concentrations in all data sets to deviate significantly from the normal distribution. Hence, further statistical analysis was performed using non-parametric tests (e.g. Wilcoxon signed rank test and Spearman rank order correlation). In all instances, where concentrations were  $\leq$  LOQ, (limit of quantification) concentrations were assumed to equal half the LOQ (Table SI-2) and *P* values  $<0.05$  were considered significant.

## Results and discussion

### Occurrence and levels of OHCs in Egyptian serum

All target PCBs, PBDEs and OCPs were detected in the analysed serum samples. A statistical summary of target OHCs concentrations in the analysed samples is provided in Table 1 (the full dataset is given in Table SI-5). *p,p'*-DDE was the only target compound to be detected in all the analysed serum samples, with the highest median concentration (131 ng/g lw, *n*=67). BDE-209 was the least frequently detected contaminant (detection frequency = 36%) with a median concentration below the method LOQ. Interestingly, the OHC contamination profile in patients and control was almost identical.  $\Sigma$ DDT (calculated as *p,p'*-DDE + *p,p'*-DDT) showed the highest median contribution to the OHC profile in serum of patients and control (77%), while oxychlordan showed the lowest contribution (1%) (Figure 1). This is not dissimilar to previous reports of OHC profiles in serum of non-occupationally exposed adults from

Romania, Belgium, Pakistan, Saudi Arabia, USA and Canada.<sup>12, 22-24</sup> Predominance of p,p'-DDE as the major OHC in serum has been attributed to a combination of factors including continuous exposure via diet or breast milk, high bioaccumulation potential and biomagnification along the food chain, in addition to long elimination half-life (median  $t_{0.5}$  of p,p'-DDE in human serum was more than 8 years).<sup>25</sup> Moreover, while DDT is listed under the Stockholm Convention on POPs, several countries in Africa, Asia and Latin America still use DDT for vector control. This can hamper the international efforts to reduce the global contamination levels with this chlorinated pesticide, due to its environmental persistence and potential for long-range atmospheric transport. In Egypt, DDT was used as a pesticide for cotton fields (1952-1971) with reported stockpiles of 600 tons.<sup>26</sup> It has been officially prohibited from agricultural use since 1980, and in 1996 a Ministerial Decree prohibited the import and use of multiple pesticides including DDT in Egypt.<sup>26</sup> While the higher levels of p,p'-DDE (the main metabolite of DDT) compared to p,p'-DDT in Egyptian serum (Table 1) is expected given its longer half-life and bioaccumulation potential, the ratio between p,p'-DDT/p,p'-DDE can be used to differentiate between current and historical exposures to this pesticide.<sup>27</sup> The low p,p'-DDT/p,p'-DDE ratio (3.7%) in this study implies high environmental persistence and bioaccumulation (c.f. recent and ongoing intake) as the major sources of DDT exposure.<sup>27</sup>

A literature survey revealed one study reporting levels of p,p'-DDE, p,p'-DDT and HCB in Egyptian colorectal cancer patients (n=31) and control (n=17) serum samples collected in 1996.<sup>28</sup> Notwithstanding the caveats related to the analytical methodology applied at this time, the median concentrations of p,p'-DDE, p,p'-DDT and HCB in the current study are approximately half of those reported in 1996.<sup>28</sup> Similarly, Tawfic et al. reported mean  $\Sigma$ PCBs concentrations of  $54 \pm 17$ ,  $59 \pm 23$  and  $61 \pm 21$  ng/g whole weight

in serum samples of Egyptian females diagnosed with invasive adenocarcinoma of the breast (n=43), suffering benign breast disease (n=21) and normal healthy females (n=11), respectively.<sup>29</sup> These levels reported in the year 2000 are substantially higher than mean  $\Sigma$ PCBs in serum of colorectal cancer patients and control group in the present study (Table 1). This is generally in agreement with the globally observed decrease in the levels of these legacy POPs in the environment and humans as a result of various legislations that ban their production and use.<sup>30</sup>

To the authors' knowledge, this is the first study to provide levels of PCP, OxC, and PBDEs in Egyptian serum or any other human matrices, which precludes any discussion of the temporal trends of these contaminants in the Egyptian population.

### **Comparison of OHC levels in serum of patients and control group**

Due to the non-parametric distribution of the generated datasets, Wilcoxon signed rank test was used to compare the differences in median and distribution between the concentrations of target OHCs in colorectal cancer patients and the control group. No statistically significant differences ( $P>0.05$ ) were observed between the levels of individual target OHCs,  $\Sigma$ PCBs,  $\Sigma$ OCPs,  $\Sigma$ PBDEs and  $\Sigma$ OHCs in serum of colorectal cancer patients and the control group (Table SI-4). The median percent contribution of the studied OHCs to the overall contamination profile in serum of patients and control group was almost identical (Figure 1). Interestingly, the mono-ortho-PCB 118 was the only investigated contaminant showing border line significant difference ( $P=0.053$ ) between its concentrations in serum of patients (median = 2.9 ng/g lw) and control group (median = 0.5 ng/g lw). This is in line with the findings of Howsam et al. who reported an elevated risk of colorectal cancer in Catalan population (n= 137, controls = 76) associated with higher serum concentrations of mono-ortho PCB congeners 28 and

118, while HCB and p,p'-DDE produced no significant increases in colorectal cancer risk.<sup>31</sup> Similarly, a previous study from Egypt reported no significant differences in levels of p,p'-DDE, p,p'-DDT and HCB, in serum of colorectal cancer patients (n= 31) compared to a healthy control group (n = 17).<sup>28</sup> However, other studies from Tunisia (n = 69, controls = 56 females) and Singapore (n = 60, controls = 60 males) have indicated positive associations between concentrations of p,p'-DDE and various PCBs in serum and the risk of breast and prostate cancers, respectively.<sup>32, 33</sup> Therefore, bearing in mind the relatively small sample number; the lack of significant differences between serum levels of OHCs in colorectal cancer patients and the control group in the current study should be interpreted with caution. More research is required on a larger sample size to confirm these findings.

### **Factors affecting the levels of OHCs in Egyptian serum**

Gender, age and BMI were investigated as potential factors influencing the levels of target OHCs in the studied serum samples. The use of other socio-economic factors (e.g. profession, location, diet, income and education) was not allowed by the Ethics Committee to maintain the anonymity of the study participants.

No statistically significant differences (Wilcoxon signed rank test,  $P > 0.05$ ) were observed between the concentrations of OHCs in the serum of males and females among the colorectal cancer patients and the control group. Therefore, we pooled the results from both males and females to study possible associations between contaminant levels in serum, age and BMI of the study participants in the patients and control group (Table 2). Results revealed a significant positive correlation between the levels of  $\Sigma$ DDT and the age of the blood donors (Table 2). Previous studies from France (n = 386, aged 18-74 years, randomly selected among the participants in the French Nutrition and Health

Survey),<sup>34</sup> Spain (n= 1259 pregnant women, aged >16 to 40 years),<sup>35</sup> USA (n=192 healthy females, aged 45 to 85 years)<sup>36</sup> and UK (n=153 healthy volunteers, aged 22-80 years)<sup>15</sup> have also reported significant increase of serum DDT concentrations with age. This can be explained by the long half-life and high bioaccumulation potential of DDT, combined with decreased metabolism and elimination of OCPs with increasing age<sup>27</sup>. Despite its lipophilic nature, published results differ with regard to the association between DDT serum levels and BMI.<sup>12</sup> While  $\Sigma$ DDT was significantly correlated with BMI in the present study, no significant association was observed in French adults (despite half the population with BMI > 25 including 15% with BMI >30).<sup>34</sup> This may be attributable to differences in exposure (e.g. diet) and socioeconomic (e.g. profession) factors influencing the serum levels of OHCs in different populations.<sup>34</sup> It's worth mentioning that the concentrations of p,p'-DDE in the present study contributed  $96 \pm 23\%$  and  $83 \pm 34\%$  to  $\Sigma$ DDT and  $\Sigma$ OCPs, respectively. Therefore, it's reasonable to conclude that the significant positive associations of serum  $\Sigma$ DDT and  $\Sigma$ OCPs with participants age and BMI (Table 2) are driven mainly by the concentrations of p,p'-DDE. Interestingly, PCP levels, the second largest contributor to serum  $\Sigma$ OCPs, showed a significant positive correlation with  $\Sigma$ DDT concentrations in both the patients and control groups (Table 2). There is no clear explanation for this strong correlation. In addition to direct exposure via diet, it has been suggested that PCP in serum can result from HCB metabolism.<sup>23</sup> Since no significant correlation was observed between the levels of  $\Sigma$ DDT and HCB or PCP and HCB in serum (Table 2), it is likely that the correlation between  $\Sigma$ DDT and PCP concentrations originate from common exposure sources to these two contaminants (e.g. diet).

PCP levels were significantly correlated to BMI in the serum of the control group. A possible explanation for this association is that BMI could alter PCP toxicokinetics and

accumulation of the compound in body fat resulting in increased internal concentration in obese individuals.<sup>37</sup> HCB levels showed a significant correlation with patients' age (Table 2), which may be attributable to bioaccumulation resulting in increased serum concentrations with age.<sup>12, 34</sup>

### Comparison with other countries

The concentrations of target OHCs in the analysed Egyptian serum samples (n=67, patients + control group) were compared to those reported previously from other countries (Figure 2 and Table SI-6). While we are aware that studies from different countries have targeted various populations (e.g. pregnant women, males, diabetic patients and general population) of different age groups with different lifestyles and habits, we believe that such comparison is beneficial to provide a general perspective of the measured concentrations of target OHCs in human blood from Egypt compared to other parts of the world. This is of particular interest given the global distribution of the studied contaminants and the synchronised international efforts required to minimize exposure and reduce human body burdens of these OHCs.

Median HCB level (5.9 ng/g lw) in Egyptian serum was at the lower end of this contaminant concentrations reported globally. This is consistent with low serum HCB concentrations in other developing countries like Saudi Arabia (*n=60, 40 diabetic patients and 20 healthy controls*),<sup>24</sup> Pakistan (*n=85, 34 mothers, 34 children (3-10 years) and 17 general population*)<sup>22</sup> and Bangladesh (*n=24 males*).<sup>38</sup> Higher serum HCB concentrations were reported from China (*n=26, median = 39 ng/g lw*)<sup>39</sup>, Spain (*n=2433, median = 34 ng/g lw*)<sup>40</sup> and USA (*n = 341 males, 15 ng/g lw*).<sup>41</sup> This may reflect less historical use of HCB in the developing countries. In Egypt, HCB was detected at concentrations of 4 - 93 ng/L in water samples collected at different locations along

the river Nile in 1995. It was also detected at low levels in Egyptian fish (2.5-20 ng/g wet weight) and vegetables (3-9 ng/g wet weight)<sup>42</sup>, indicating dietary intake as the predominant source of human exposure to HCB.

PCBs in Egyptian serum showed a similar profile to that reported from other countries with PCB 138 as the predominant congener (median = 4.93 ng/g lw) followed by PCB 153 (median = 2.83 ng/g lw). However, median serum  $\Sigma$ PCBs in this study (10.36 ng/g lw) was lower than those reported elsewhere (Figure 2). This is generally in line with low PCB residues found in the Egyptian terrestrial and aquatic environment, which was attributed to the low industrial profile of the country until the 1970s.<sup>43</sup>

Unlike PCBs, the median serum concentration of p,p'-DDE in the current study (131 ng/g lw) was higher than those reported in blood samples from the general population of UK (n=153, 100 ng/g lw)<sup>15</sup>, Belgium (n=204, 64 ng/g lw)<sup>12</sup> and Canada (n= 5000, 63 ng/g lw).<sup>12</sup> DDT was never used for Malaria control in Egypt. However, it was widely applied to treat cotton fields from 1952 to 1971. In the 1970s, DDT usage decreased gradually due to the resistance developed by the cotton leafworm, *Spodoptera littoralis*, until it was officially prohibited from all agricultural applications in 1980.<sup>26</sup> DDTs (dominated by p,p'-DDE) were detected in Egyptian fresh water (3-1048 ng/L), vegetables (1.78-2.82 ng/g), meat (67 ng/g fat) and fish (8-91 ng/g wet weight) products, indicating widespread contamination of the Egyptian environment with DDT.<sup>42</sup>

Fewer studies have reported on the levels of PCP and oxychlordan in serum from different countries. The median serum level of PCP in the current study (229 pg/mL) was lower than those reported in serum samples of pregnant women from the Netherlands (n=69, median = 970 pg/mL) and Sweden (n=15, 2830 pg/mL)<sup>44</sup> as well as



Norwegian women ( $n=281$ , 771 pg/mL)<sup>9</sup>, but higher than the concentrations reported from Japan ( $n = 20$  males, 140 pg/mL)<sup>45</sup> and Pakistan (130 pg/mL).<sup>22</sup>

OxC is the oxidised, relatively more polar metabolite of the pesticide Chlordane, which is more likely to be detected in serum. Median level of OxC in Egyptian serum was below LOQ, ( $<1.5$  ng/g lw) with a detection frequency of 39%. This is consistent with the very low detection frequency of Chlordane in the Egyptian environment.<sup>42</sup>

To the authors' knowledge, this is the first study to report PBDE levels in Egyptian serum. BDE-47 was the most frequently detected congener (87%) with the highest median level (0.97 ng/g lw), followed by BDE-99 (77%, 0.61 ng/g lw). This is consistent with previous studies from other countries, indicating exposure to the penta-BDE commercial formulation.<sup>46</sup> Median penta-BDE level (1.9 ng/g lw, sum of congeners 47, 99, 100, 153 and 154) in Egyptian serum was at the lower end of those reported from other countries (Figure 2). A recent study reported low concentrations of PBDEs in dust samples collected from Egyptian homes ( $n=17$ , median penta-BDE = 5.2 ng/g), workplaces ( $n=9$ , 11.6 ng/g) and cars ( $n=5$ , 35.6 ng/g).<sup>47</sup> Indoor dust was highlighted as the major pathway of human exposure to PBDEs via ingestion and dermal contact.<sup>48</sup> Therefore, the low levels of PBDEs in Egyptian serum samples can be explained by the low concentrations of these flame retardants in Egyptian indoor dust.

BDE-209 (the major congener of the deca-BDE mixture) and BDE-183 (the indicator congener for octa-BDE mixture) were detected in Egyptian serum at lower frequency and concentrations than the penta-BDE congeners (Table 1). This may be attributed to the high lipophilicity and molecular weight of these congeners, which is likely to reduce their bioavailability to humans following exposure via indoor dust or diet. Moreover, the short serum half-life of BDE-209 (7-14 days) indicates that the detected levels in serum likely reflect recent exposure of the participants, rather than prolonged chronic

exposure.<sup>49</sup> Overall, the concentrations of PBDEs detected in Egyptian serum in this study are among the lowest reported worldwide.

### **Limitations and strengths**

The current study doesn't aim and can't qualify to be an epidemiological case-control study. This is mainly due to the ethical restrictions which limited the public disclosure of risk factors to age, gender, and BMI; excluding other known important factors to ensure participant anonymity. In addition, the relatively small sample size combined with the 3 risk factors studied can only detect odds ratios of 4 ~5 (or higher) with sufficient statistical power. Uncertainties in the study also include the degree of representability of the studied samples to the general population given the overall small number of participants and the relatively high average BMI observed for both the patients and control group (Table 1).

However, this work provides the first human biomonitoring data of various OHCs in the Egyptian population, which significantly augments the current knowledge about the global distribution of these contaminants. Furthermore, due to the general lack of statistically significant differences between the patients and control group, the current paper focuses on characterising the levels and profiles of a broad suite of OHCs in Egyptian blood in comparison to previous reports from other countries and regions. This provides novel insights into the internal human exposure to contaminants like PBDEs and other OCPs in Egypt, which may trigger further regulatory and/or socioeconomic actions to reduce human exposure to these hazardous chemicals in the developing countries.

## **Acknowledgement**

The contribution of AC has received funding from the European Union Seventh Framework Programme [FP7/2007-2013] within the INTERFLAME grant agreement no. 295138. The authors gratefully acknowledge Wesam M. El-Koussi; Sally A. El Zohny and Aml I. Mekawy for their help during sample preparation and analysis.

## **Supplementary data**

Further details of the analytical methodology, quality assurance/quality control parameters and concentrations of all target compounds in individual serum samples are available as supplementary data.

## References

1. UNEP, United Nations Environment Programme, Persistent Organic Pollutants Review Committee (POPRC), Reports and Decisions. <http://chm.pops.int/TheConvention/POPsReviewCommittee/ReportsandDecisions> **2009**.
2. Xu, W.; Wang, X.; Cai, Z., Analytical chemistry of the persistent organic pollutants identified in the Stockholm Convention: A review. *Anal Chim Acta* **2013**, *790*, 1-13.
3. Pi, N.; Chia, S. E.; Ong, C. N.; Kelly, B. C., Associations of serum organohalogen levels and prostate cancer risk: Results from a case-control study in Singapore. *Chemosphere* **2016**, *144*, 1505-12.
4. Kuo, C.-C.; Moon, K.; Thayer, K. A.; Navas-Acien, A., Environmental Chemicals and Type 2 Diabetes: An Updated Systematic Review of the Epidemiologic Evidence. *Curr Diab Rep* **2013**, *13*, (6), 831-849.
5. Gascon, M.; Morales, E.; Sunyer, J.; Vrijheid, M., Effects of persistent organic pollutants on the developing respiratory and immune systems: A systematic review. *Environ Int* **2013**, *52*, 51-65.
6. Darnerud, P. O., Toxic effects of brominated flame retardants in man and in wildlife. *Environ Int* **2003**, *29*, (6), 841-53.
7. Law, R. J.; Covaci, A.; Harrad, S.; Herzke, D.; Abdallah, M. A. E.; Femie, K.; Toms, L.-M. L.; Takigami, H., Levels and trends of PBDEs and HBCDs in the global environment: Status at the end of 2012. *Environ Int* **2014**, *65*, 147-158.
8. Bjermo, H.; Darnerud, P. O.; Lignell, S.; Pearson, M.; Rantakokko, P.; Nalsen, C.; Barbieri, H. E.; Kiviranta, H.; Lindroos, A. K.; Glynn, A., Fish intake and breastfeeding time are associated with serum concentrations of organochlorines in a Swedish population. *Environ Int* **2013**, *51*, 88-96.

9. Rylander, C.; Lund, E.; Froyland, L.; Sandanger, T. M., Predictors of PCP, OH-PCBs, PCBs and chlorinated pesticides in a general female Norwegian population. *Environ Int* **2012**, *43*, 13-20.
10. Whitehead, T. P.; Smith, S. C.; Park, J.-S.; Petreas, M. X.; Rappaport, S. M.; Metayer, C., Concentrations of persistent organic pollutants in California women's serum and residential dust. *Environ Res* **2015**, *136*, 57-66.
11. Abdallah, M. A. E.; Pawar, G.; Harrad, S., Evaluation of in vitro vs. in vivo methods for assessment of dermal absorption of organic flame retardants: A review. *Environ Int* **2015**, *74*, 13-22.
12. Aylward, L. L.; Green, E.; Porta, M.; Toms, L.-M.; Den Hond, E.; Schulz, C.; Gasull, M.; Pumarega, J.; Conrad, A.; Kolossa-Gehring, M.; Schoeters, G.; Mueller, J. F., Population variation in biomonitoring data for persistent organic pollutants (POPs): An examination of multiple population-based datasets for application to Australian pooled biomonitoring data. *Environ Int* **2014**, *68*, 127-138.
13. Rawn, D. F. K.; Ryan, J. J.; Sadler, A. R.; Sun, W.-F.; Haines, D.; Macey, K.; Van Oostdam, J., PCDD/F and PCB concentrations in sera from the Canadian Health Measures Survey (CHMS) from 2007 to 2009. *Environ Int* **2012**, *47*, 48-55.
14. Rawn, D. F. K.; Ryan, J. J.; Sadler, A. R.; Sun, W.-F.; Weber, D.; Laffey, P.; Haines, D.; Macey, K.; Van Oostdam, J., Brominated flame retardant concentrations in sera from the Canadian Health Measures Survey (CHMS) from 2007 to 2009. *Environ Int* **2014**, *63*, 26-34.
15. Thomas, G. O.; Wilkinson, M.; Hodson, S.; Jones, K. C., Organohalogen chemicals in human blood from the United Kingdom. *Environ Poll* **2006**, *141*, (1), 30-41.
16. Gado, A.; Ebeid, B.; Abdelmohsen, A.; Axon, A., Colorectal cancer in Egypt is commoner in young people: Is this cause for alarm? *Alex J Med* **2014**, *50*, (3), 197-201.

17. Veruttipong, D.; Soliman, A. S.; Gilbert, S. F.; Blachley, T. S.; Hablas, A.; Ramadan, M.; Rozek, L. S.; Seifeldin, I. A., Age distribution, polyps and rectal cancer in the Egyptian population-based cancer registry. *World J Gastroenterol* **2012**, *18*, (30), 3997-4003.
18. Abdel-Rahman, S. Z.; Soliman, A. S.; Bondy, M. L.; Omar, S.; El-Badawy, S. A.; Khaled, H. M.; Seifeldin, I. A.; Levin, B., Inheritance of the 194Trp and the 399Gln variant alleles of the DNA repair gene XRCC1 are associated with increased risk of early-onset colorectal carcinoma in Egypt. *Cancer Lett* **2000**, *159*, (1), 79-86.
19. Dey, S.; Zhang, Z. Z.; Hablas, A.; Seifeldein, I. A.; Ramadan, M.; El-Hamzawy, H.; Soliman, A. S., Geographic patterns of cancer in the population-based registry of Egypt: Possible links to environmental exposures. *Cancer Epidemiol* **2011**, *35*, (3), 254-264.
20. Mahfouz, E. M.; Sadek, R. R.; Abdel-Latief, W. M.; Mosallem, F. A. H.; Hassan, E. E., The role of dietary and lifestyle factors in the development of colorectal cancer: case control study in Minia, Egypt. *Cent Eur J Public Health* **2014**, *22*, (4), 215-222.
21. Phillips, D. L.; Pirkle, J. L.; Burse, V. W.; Bernert, J. T., Jr.; Henderson, L. O.; Needham, L. L., Chlorinated hydrocarbon levels in human serum: effects of fasting and feeding. *Arch Environ Contam Toxicol* **1989**, *18*, (4), 495-500.
22. Ali, N.; Eqani, S. A.; Malik, R. N.; Neels, H.; Covaci, A., Organohalogenated contaminants (OHCs) in human serum of mothers and children from Pakistan with urban and rural residential settings. *Sci Total Environ* **2013**, *461-462*, 655-62.
23. Dirtu, A. C.; Jaspers, V. L. B.; Cernat, R.; Neels, H.; Covaci, A., Distribution of PCBs, Their Hydroxylated Metabolites, and Other Phenolic Contaminants in Human Serum from Two European Countries. *Environ Sci Technol* **2010**, *44*, (8), 2876-2883.
24. Ali, N.; Rajeh, N.; Wang, W.; Abualnaja, K. O.; Kumosani, T. A.; Albar, H. M. S.; Eqani, S. A. M. A. S.; Ismail, I. M. I., Organohalogenated contaminants in type 2 diabetic serum from Jeddah, Saudi Arabia. *Environ Poll* **2016**, *213*, 206-212.

25. Jaga, K.; Dharmani, C., Global surveillance of DDT and DDE levels in human tissues. *Int J Occup Med Environ Health* **2003**, *16*, (1), 7-20.
26. El-Shahawy, A.; Simeonov, L. I., Environmental and Health Situation with Obsolete Pesticides in Egypt. In *Environmental Security Assessment and Management of Obsolete Pesticides in Southeast Europe*, Simeonov, I. L.; Macaev, Z. F.; Simeonova, G. B., Eds. Springer Netherlands: Dordrecht, 2013; pp 209-218.
27. Ahlborg, U. G.; Lipworth, L.; Titus-Ernstoff, L.; Hsieh, C. C.; Hanberg, A.; Baron, J.; Trichopoulos, D.; Adami, H. O., Organochlorine compounds in relation to breast cancer, endometrial cancer, and endometriosis: an assessment of the biological and epidemiological evidence. *Crit Rev Toxicol* **1995**, *25*, (6), 463-531.
28. Soliman, A. S.; Smith, M. A.; Cooper, S. P.; Ismail, K.; Khaled, H.; Ismail, S.; McPherson, R. S.; Seifeldin, I. A.; Bondy, M. L., Serum organochlorine pesticide levels in patients with colorectal cancer in Egypt. *Arch Environ Health* **1997**, *52*, (6), 409-415.
29. Ahmed, M. T.; Loutfy, N.; El Shiekh, E., Residue levels of DDE and PCBs in the blood serum of women in the Port Said region of Egypt. *J Hazard Mater* **2002**, *89*, (1), 41-48.
30. Abdallah, M. A.-E., Persistent Organic Pollutants. In *Still Only One Earth: Progress in the 40 Years Since the First UN Conference on the Environment*, The Royal Society of Chemistry: 2015; pp 150-186.
31. Howsam, M.; Grimalt, J. O.; Guino, E.; Navarro, M.; Marti-Rague, J.; Peinado, M. A.; Capella, G.; Moreno, V.; Bellvitge Colorectal Canc, G., Organochlorine exposure and colorectal cancer risk. *Environ Health Perspect* **2004**, *112*, (15), 1460-1466.
32. Arrebola, J. P.; Belhassen, H.; Artacho-Cordon, F.; Ghali, R.; Ghorbel, H.; Boussen, H.; Perez-Carrascosa, F. M.; Exposito, J.; Hedhili, A.; Olea, N., Risk of female breast cancer and serum concentrations of organochlorine pesticides and polychlorinated biphenyls: A case-control study in Tunisia. *Sci Total Environ* **2015**, *520*, 106-113.

33. Pi, N.; Chia, S. E.; Ong, C. N.; Kelly, B. C., Associations of serum organohalogen levels and prostate cancer risk: Results from a case-control study in Singapore. *Chemosphere* **2016**, *144*, 1505-1512.
34. Saoudi, A.; Frery, N.; Zeghnoun, A.; Bidondo, M.-L.; Deschamps, V.; Goeen, T.; Gamier, R.; Guldner, L., Serum levels of organochlorine pesticides in the French adult population: The French National Nutrition and Health Study (ENNS), 2006-2007. *Sci Total Environ* **2014**, *472*, 1089-1099.
35. Ibarluzea, J.; Alvarez-Pedrerol, M.; Guxens, M.; Marina, L. S.; Basterrechea, M.; Lertxundi, A.; Etxeandia, A.; Goni, F.; Vioque, J.; Ballester, F.; Sunyer, J.; Project, I., Sociodemographic, reproductive and dietary predictors of organochlorine compounds levels in pregnant women in Spain. *Chemosphere* **2011**, *82*, (1), 114-120.
36. Moysich, K. B.; Ambrosone, C. B.; Mendola, P.; Kostyniak, P. J.; Greizerstein, H. B.; Vena, J. E.; Menezes, R. J.; Swede, H.; Shields, P. G.; Freudenheim, J. L., Exposures associated with serum organochlorine levels among postmenopausal women from western New York State. *Am J Ind Med* **2002**, *41*, (2), 102-110.
37. Zheng, W.; Wang, X.; Yu, H.; Tao, X.; Zhou, Y.; Qu, W., Global trends and diversity in pentachlorophenol levels in the environment and in humans: a meta-analysis. *Environ Sci Technol* **2011**, *45*, (11), 4668-75.
38. Mamun, M.; Nahar, N.; Mosihuzzaman, M.; Linderholm, L.; Athanasiadou, M.; Bergman, A., Traditional organochlorine pollutants in blood from humans living in the Bangladesh capital area. *Organohalog. Compd.* **2007**, *69*, 2026-2030.
39. Bi, X.; Thomas, G. O.; Jones, K. C.; Qu, W.; Sheng, G.; Martin, F. L.; Fu, J., Exposure of electronics dismantling workers to polybrominated diphenyl ethers, polychlorinated biphenyls, and organochlorine pesticides in South China. *Environ Sci Technol* **2007**, *41*, (16), 5647-53.



40. Arrebola, J. P.; Ocana-Riola, R.; Arrebola-Moreno, A. L.; Fernandez-Rodriguez, M.; Martin-Olmedo, P.; Fernandez, M. F.; Olea, N., Associations of accumulated exposure to persistent organic pollutants with serum lipids and obesity in an adult cohort from Southern Spain. *Environ Poll* **2014**, *195*, 9-15.
41. Meeker, J. D.; Altshul, L.; Hauser, R., Serum PCBs, p,p'-DDE and HCB predict thyroid hormone levels in men. *Environ Res* **2007**, *104*, (2), 296-304.
42. Mansour, S. A., Environmental Impact of Pesticides in Egypt. *Rev Environ Contam T* **2008**, *196*, 1-51.
43. Ahmed, M. T., Persistent Organic Pollutants in Egypt - an Overview. *Nato Sci S Ss Iv Ear* **2006**, *69*, 25-38.
44. Meijer, L.; Weiss, J.; Van Velzen, M.; Brouwer, A.; Bergman, A.; Sauerf, P. J. J., Serum concentrations of neutral and phenolic organohalogens in pregnant women and some of their infants in the Netherlands. *Environ Sci Technol* **2008**, *42*, (9), 3428-3433.
45. Fujii, Y.; Harada, K. H.; Hitomi, T.; Kobayashi, H.; Koizumi, A.; Haraguchi, K., Temporal trend and age-dependent serum concentration of phenolic organohalogen contaminants in Japanese men during 1989-2010. *Environ Poll* **2014**, *185*, 228-233.
46. Frederiksen, M.; Vorkamp, K.; Thomsen, M.; Knudsen, L. E., Human internal and external exposure to PBDEs--a review of levels and sources. *Int J Hyg Environ Health* **2009**, *212*, (2), 109-34.
47. Hassan, Y.; Shoeib, T., Levels of polybrominated diphenyl ethers and novel flame retardants in microenvironment dust from Egypt: An assessment of human exposure. *Sci Total Environ* **2015**, *505*, 47-55.
48. Trudel, D.; Scheringer, M.; von Goetz, N.; Hungerbuehler, K., Total Consumer Exposure to Polybrominated Diphenyl Ethers in North America and Europe. *Environ Sci Technol* **2011**, *45*, (6), 2391-2397.

49. Abdallah, M. A.; Harrad, S., Polybrominated diphenyl ethers in UK human milk: Implications for infant exposure and relationship to external exposure. *Environ Int* **2014**, *63*, 130-136.

ACCEPTED MANUSCRIPT

## Tables

**Table 1:** Statistical summary of the concentrations (ng/g lipid weight) of target OHCs in Egyptian serum samples.

	Patients (n=35, 19 males and 16 females)						Control (n=32, 17 males and 15 females)					
	DF* (%)	Median	Average	SD	Min	Max	DF (%)	Median	Average	SD	Min	Max
<b>Age</b>		47	48	9	34	62		43	44	9	31	61
<b>BMI</b>		29	31	6	23	43		32	33	7	19	48
<b>PCB-118</b>	70	1.34	1.91	1.92	<LOQ	6.88	69	0.50	1.52	1.82	<LOQ	6.10
<b>PCB-138</b>	91	4.93	5.41	3.30	<LOQ	12.45	94	5.00	4.92	3.03	<LOQ	12.86
<b>PCB-153</b>	80	2.66	3.01	2.50	<LOQ	10.17	66	3.03	3.11	2.59	<LOQ	8.03
<b>PCB-170</b>	60	0.48	0.78	0.99	<LOQ	4.66	63	0.52	0.65	0.80	<LOQ	4.36
<b>PCB-180</b>	57	0.27	0.57	0.76	<LOQ	3.14	56	0.33	0.58	0.69	<LOQ	3.35
<b>ΣPCBs</b>		<b>10.17</b>	<b>11.67</b>	<b>6.22</b>	<b>&lt;LOQ</b>	<b>28.63</b>		<b>10.76</b>	<b>10.78</b>	<b>5.45</b>	<b>&lt;LOQ</b>	<b>21.94</b>
<b>pp-DDE</b>	100	126.26	230.95	275.21	8.44	1061.65	100	139.75	184.98	205.39	12.18	930.40
<b>pp-DDT</b>	34	<LOQ	8.49	10.35	<LOQ	47.65	53	4.58	8.31	7.17	3.14	33.56
<b>ΣDDT</b>		<b>133.97</b>	<b>239.44</b>	<b>281.40</b>	<b>12.44</b>	<b>1109.29</b>		<b>145.24</b>	<b>193.29</b>	<b>212.21</b>	<b>16.18</b>	<b>960.11</b>
<b>HCB</b>	86	5.87	6.37	3.85	<LOQ	14.04	81	6.41	6.60	4.81	<LOQ	18.34
<b>OxC</b>	46	1.50	2.10	0.93	<LOQ	5.28	31	1.50	1.87	0.89	<LOQ	5.12
<b>PCP</b>	89	17.99	33.26	45.87	<LOQ	192.51	94	15.97	28.73	35.48	<LOQ	142.54
<b>ΣOCPs</b>		<b>155.85</b>	<b>281.17</b>	<b>314.65</b>	<b>25.01</b>	<b>1279.01</b>		<b>168.40</b>	<b>230.48</b>	<b>244.42</b>	<b>28.88</b>	<b>1114.83</b>
<b>BDE-47</b>	86	0.88	1.65	1.95	<LOQ	7.33	88	1.13	1.89	2.19	<LOQ	8.31
<b>BDE-100</b>	57	<LOQ	0.56	0.85	<LOQ	3.29	66	0.19	0.52	0.68	<LOQ	2.43
<b>BDE-99</b>	80	0.50	1.30	1.63	<LOQ	6.11	75	0.71	1.40	1.66	<LOQ	6.81
<b>BDE-154</b>	34	<LOQ	0.38	0.72	<LOQ	3.02	50	0.04	0.25	0.44	<LOQ	2.23

<b>BDE-153</b>	54	0.11	0.53	0.89	<LOQ	3.78	69	0.20	0.44	0.61	<LOQ	2.80
<b>BDE-183</b>	57	0.29	0.52	0.64	<LOQ	2.22	56	0.31	0.57	0.67	<LOQ	2.08
<b>BDE-209</b>	34	<LOQ	4.03	6.39	<LOQ	21.39	41	<LOQ	4.62	7.05	<LOQ	25.40
<b>ΣPBDEs</b>		<b>5.95</b>	<b>8.96</b>	<b>7.98</b>	<b>&lt;LOQ</b>	<b>25.99</b>		<b>7.84</b>	<b>9.69</b>	<b>8.78</b>	<b>&lt;LOQ</b>	<b>41.17</b>

\* Detection frequency.

**Table 2:** Spearman rank order correlations among serum concentrations of OHCs, age and BMI of the study participants.

Patients (n=35)		Age	BMI	ΣPCBs**	ΣDDT <sup>#</sup>	HCB	OxC	PCP	ΣOCPs <sup>\$</sup>
BMI	R	0.223							
	P	0.197							
ΣPCBs	R	0.025	-0.029						
	P	0.889	0.868						
ΣDDT	R	<b>0.349*</b>	<b>0.343*</b>	0.089					
	P	<b>0.04</b>	<b>0.044</b>	0.613					
HCB	R	<b>0.386*</b>	-0.015	0.157	0.089				
	P	<b>0.022</b>	0.931	0.367	0.611				
OxC	R	-0.04	-0.246	-0.071	0.129	-0.027			
	P	0.818	0.154	0.687	0.461	0.877			
PCP	R	0.038	0.265	0.007	<b>0.463*</b>	-0.198	0.278		
	P	0.83	0.124	0.966	<b>0.005</b>	0.254	0.107		
ΣOCPs	R	0.323	<b>0.348*</b>	0.085	<b>0.989*</b>	0.098	0.16	<b>0.549*</b>	
	P	0.058	<b>0.04</b>	0.626	<b>0.001</b>	0.576	0.357	<b>0.001</b>	
ΣPBDEs <sup>+</sup>	R	0.054	0.062	-0.206	-0.084	0.191	-0.143	-0.149	-0.141
	P	0.759	0.723	0.236	0.63	0.272	0.418	0.421	0.419
Control (n=32)		Age	BMI	ΣPCBs	ΣDDT	HCB	OxC	PCP	ΣOCPs
BMI	R	0.321							
	P	0.073							
ΣPCBs	R	0.058	0.175						
	P	0.751	0.339						
ΣDDT	R	<b>0.399*</b>	<b>0.437*</b>	0.023					
	P	<b>0.024</b>	<b>0.012</b>	0.901					
HCB	R	0.233	0.163	0.146	-0.031				
	P	0.199	0.373	0.425	0.865				
OxC	R	0.198	-0.068	0.275	0.214	-0.211			
	P	0.276	0.712	0.127	0.239	0.247			
PCP	R	0.177	<b>0.370*</b>	0.021	<b>0.694*</b>	0.038	0.174		
	P	0.331	<b>0.037</b>	0.909	<b>0.001</b>	0.836	0.342		
ΣOCPs	R	<b>0.407*</b>	<b>0.480*</b>	0.042	<b>0.991*</b>	0.018	0.173	<b>0.736*</b>	
	P	<b>0.021</b>	<b>0.005</b>	0.820	<b>0.001</b>	0.923	0.344	<b>0.001</b>	
ΣPBDEs	R	-0.341	-0.124	-0.254	-0.115	-0.291	-0.058	0.124	-0.123
	P	0.056	0.499	0.160	0.530	0.106	0.753	0.500	0.503

\* Statistically significant at the 0.05 level.

\*\* Sum of PCB congeners 118, 138, 153, 170 and 180.

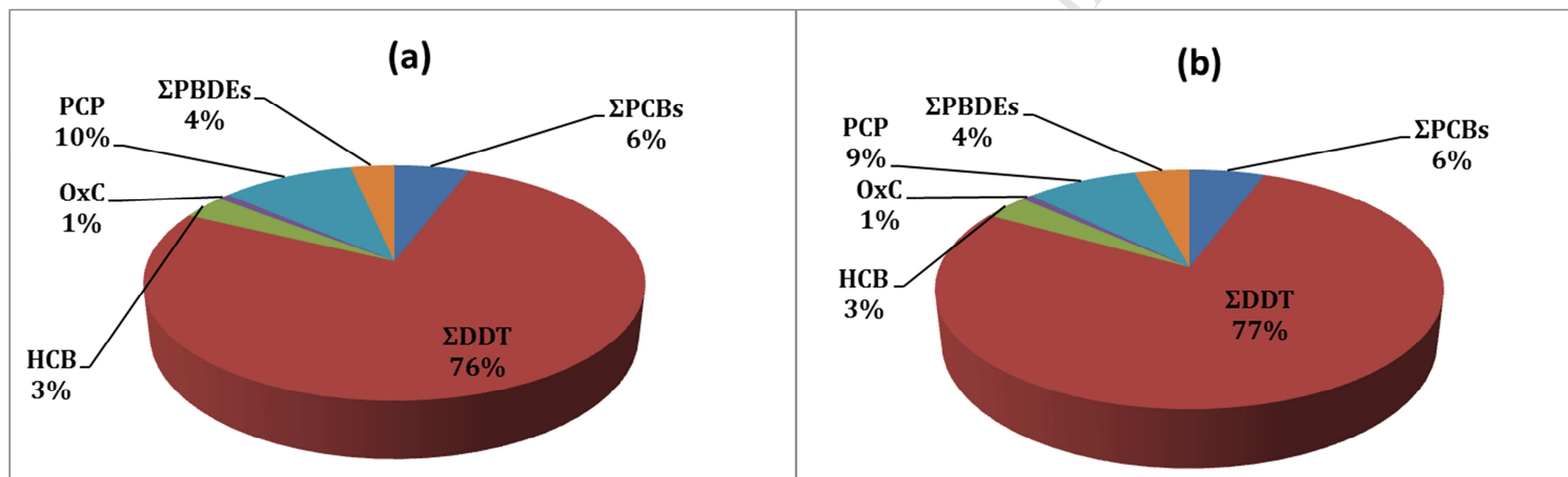
<sup>#</sup> Sum of p,p'-DDE and p,p'-DDT.

<sup>\$</sup> Sum of p,p'-DDE, p,p'-DDT, HCB, OxC and PCP.

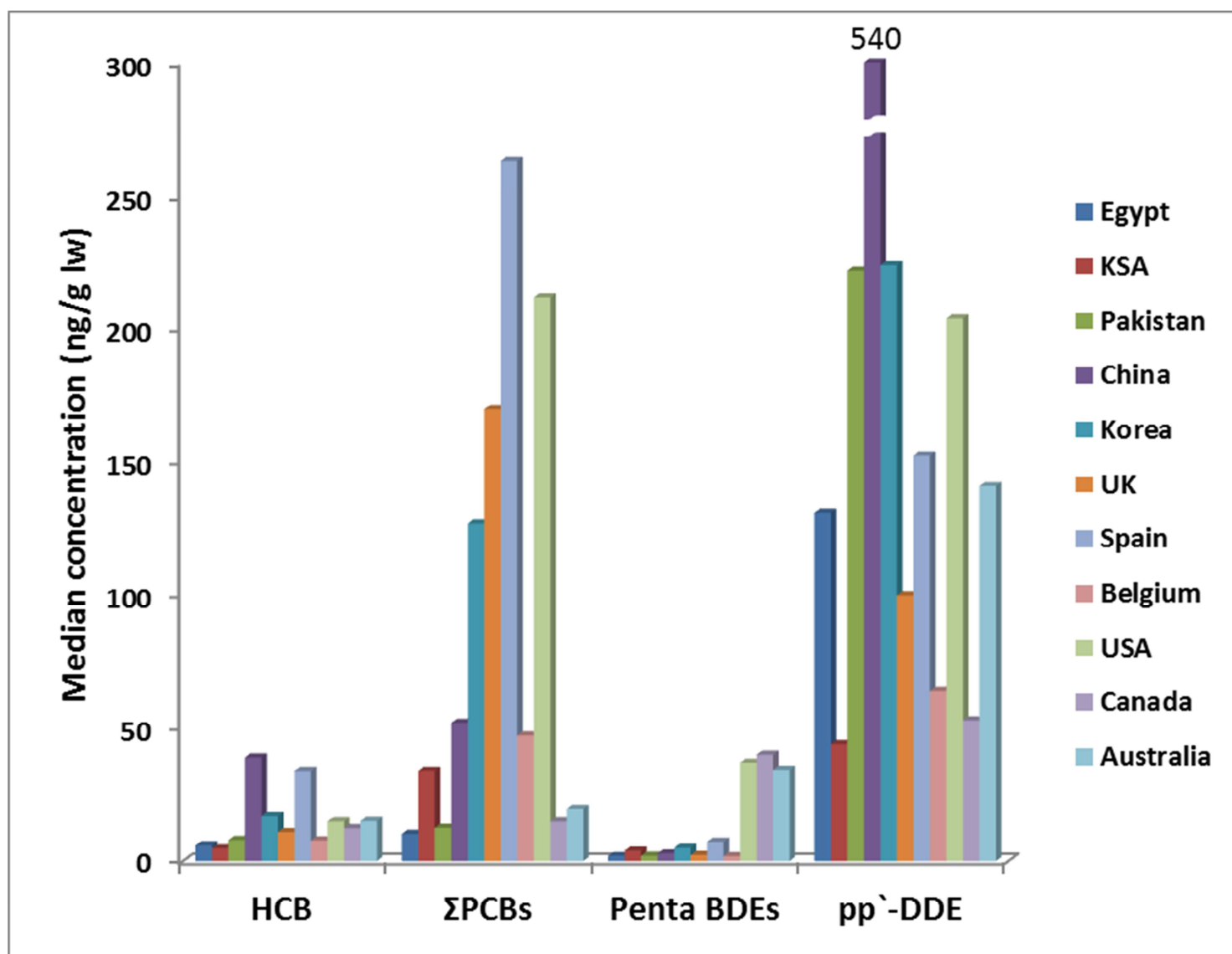
<sup>+</sup> Sum of PBDE congeners 47, 99, 100, 153, 154, 183 and 209.

## Figures

**Figure 1:** Median profile of the studied OHCs in serum samples of (a) colorectal cancer patients (n=35) and (b) control group (n=32).



**Figure 2:** Median concentrations (ng/g lw) of HCB,  $\Sigma$ PCBs, Prnta-BDEs and p,p'-DDE in human serum samples from different countries.



## Research Highlights

---

- PBDEs, PCBs and OCPs were determined in 67 human blood samples from Egypt
- No significant differences between colorectal cancer patients and control group
- $\Sigma$ DDT and  $\Sigma$ OCPs increased significantly with age and BMI
- PBDEs and PCBs in serum of Egyptians are among the lowest worldwide